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Metabolic Syndrome and Discrepancy between Actual and Self-Identified Good Weight: Aerobic Center Longitudinal Study

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Abstract

This study examined whether the discrepancy between measured and self-identified good weight (weight discrepancy) predicts metabolic syndrome (MetSyn). This study included 6,413 participants enrolled in the Aerobics Center Longitudinal Study (mean follow-up: 4.8 ± 3.8 years). Weight discrepancy was defined as measured weight minus self-identified good weight. MetSyn was defined using standard definitions. Hazard ratios (HRs) and 95% confidence intervals (95% CIs) for incident MetSyn, by weight discrepancy category, were estimated using Cox proportional hazards regression. The multivariable-adjusted HR for MetSyn was 3.48 (95% CI = 2.48–4.86) for those who maintained higher weight discrepancy over time compared to individuals with lower weight discrepancy. Additional adjustment for body mass index did not change this interpretation (HR = 3.44; 95% CI = 2.46–4.82). Weight discrepancy may be a useful screening characteristic

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Conflict of Interest

The authors have no conflicts of interest to declare.

and target for future interventions to further reduce the risk of chronic weight-related disorders, included MetSyn.

Keywords

Weight Discrepancy; Metabolic Syndrome; Obesity; Physical Activity

Introduction

Previous cross-sectional research has indicated that larger discrepancies between measured and self-identified good weight (i.e., weight discrepancy) have been associated with greater dieting frequency, increased snacking, yo-yo dieting, physical inactivity, tobacco use, and alcohol consumption (Blake et al., 2013). Individuals with chronically larger weight discrepancies had three times greater risk of developing type 2 diabetes compared to individuals with smaller weight discrepancies (Wirth, Blake, Hebert, Sui, & Blair, 2014). Although this concept of weight discrepancy has not been validated against measures of weight dissatisfaction, similarities do exist. For example, weight dissatisfaction also has been associated with poor eating habits (e.g., vomiting, binging), increased tobacco and alcohol consumption, diagnosed mental illness, poorer self-perceived health, and stress (Forrester-Knauss & Zemp Stutz, 2012; Garber, Boyer, Pollack, Chang, & Shafer, 2008; Keel, Baxter, Heatherton, & Joiner, 2007; Wade, Zhu, & Martin, 2011).

Although there is little research focusing on weight discrepancy and health outcomes, it is possible that the mechanisms of disease progression among those with high weight discrepancy may be similar to those proposed for high weight dissatisfaction. Those with chronically high weight discrepancies over time may experience excessive stress or negative affect, which can potentially induce numerous physiological changes (e.g., immune, metabolic, inflammatory, and behavioral changes) potentially increasing the risk of chronic disease. A similar mechanism has been proposed for weight dissatisfaction (Cernelic-Bizjak & Jenko-Praznikar, 2014; Muennig, 2008; Steptoe & Brydon, 2009) and may be applicable for weight discrepancy as well.

Although related to diabetes, metabolic syndrome (MetSyn) includes multiple factors associated with poor health characteristics (i.e., abdominal obesity, hypertension, glucose intolerance, elevated triglycerides and high-density lipoprotein cholesterol [HDL-C]) (Grundy, Brewer, Cleeman, Smith, & Lenfant, 2004). The estimated prevalence of MetSyn typically falls between 20%–30% for most countries, with an elevated prevalence mirroring a high rate of obesity in the United States, where about 69% of the population is overweight (34%) or obese (35%) (Grundy, 2008; Ogden, Carroll, Kit, & Flegal, 2014). Interventions targeting poor health behaviors, such as those associated with factors linked to the weight discrepancy, have been found to reduce the risk of MetSyn (Yamaoka & Tango, 2012). Therefore, we hypothesized that larger weight discrepancies at baseline would be associated with increased MetSyn among a population of adults from the Aerobics Center Longitudinal Study (ACLS). Additionally, we hypothesized that those with chronically higher weight

discrepancies over a period ≥ 2 years will have increased risk of MetSyn compared to those who maintained lower weight discrepancies.

Method

Participants

The ACLS enrolled volunteer patients who were referred by doctors or healthcare providers for preventive medical examinations from the Cooper Clinic (Dallas, Texas). The Cooper Institute Institutional Review Board provided annual protocol review (Blair, Kohl, et al., 1989). For these analyses, males or females needed to be ≥ 20 years old; have undergone ≥ 2 clinical examinations between 1986 and 2006; have complete data on all MetSyn components; had objectively measured weight; provided self-identified good weight and data on selected covariates; and have no baseline MetSyn, diabetes, cardiovascular disease (CVD), cancer, ulcers, gallbladder disease, jaundice, hepatitis, cirrhosis, or colon polyps. We further excluded those whose body mass index [BMI = weight(kg)/height(m)^2] did not fall between 18.5 and 50kg/m^2, as values outside this range may represent subclinical disease.

Procedure

The protocol, including all clinical and physical activity measures, have previously been described in detail and followed a standard manual of operations (Blair, Kannel, Kohl, Goodyear, & Wilson, 1989).Participants provided informed consent and arrived for the clinical examination after ≥ 12-hour fast. Information collected included personal and family health histories, fasting blood chemistry analyses, anthropometry, resting blood pressure, electrocardiogram, and a maximal graded exercise test. BMI was computed from measured weight and height.

Measures

Weight discrepancy—Weight discrepancy was defined as measured weight minus self-identified good weight, which was obtained by asking participants “What do you consider a good weight for yourself?” High weight discrepancy was defined as having a difference in measured and self-identified good weight that was above the median (males: 2.72kg; females: 3.52kg) and low weight discrepancy was defined as a difference at or below the median. Only 5% of the study population had a negative weight discrepancy that was below −2.3kg (−5 pounds). Considering that these individuals with a weight discrepancy indicating they may want to weigh more (i.e., negative values) were low in frequency and may be different than those who want to weigh less, they were removed from the analyses. Secondary analyses examined the difference in weight discrepancy using the baseline visit and the visit at which ≥ 2 years of follow-up occurred (referred to as time point two) and required that participants had at least a third follow-up visit. For secondary analyses, a four-level weight discrepancy variable was created. A participant having low or high discrepancy at both baseline and time point two was classified as ‘stayed low’ or ‘stayed high’, respectively. If a participant changed from high to low or low to high, he/she was classified as ‘became low’ or ‘became high’, respectively.

Metabolic syndrome—MetSyn criteria were based on the National Cholesterol Education Program Adult Treatment Panel guidelines with modifications from the American Heart Association and the National Heart, Lung, and Blood Institute (Grundy et al., 2004). MetSyn was defined as exceeding the cut-point values for ≥ 3 of the following components: waist circumference (males: ≥ 102cm; females: ≥ 88cm), blood pressure (systolic: ≥ 130mmHg; diastolic: ≥ 85mmHg), fasting HDL-C (males: < 40mg/dL; females: < 50mg/dL), fasting triglycerides (≥ 150mg/dL), or fasting glucose intolerance (≥ 100mg/dL).

Covariate data—A standardized medical history questionnaire was used to obtain information on smoking habits, alcohol intake, personal history of chronic disease (e.g., myocardial infarction, stroke, cancer, hypertension, diabetes, hypercholesterolemia, colon polyps) and eating habits. Physical activity was self-assessed by answering questions on current moderate and vigorous physical activity and intention regarding future activity (Blair, Kohl, et al., 1989; Lee et al., 2012), as well as objectively measured through a maximal treadmill test, which provided estimates of cardiorespiratory fitness, using a modified Balke protocol (Balke & Ware, 1959; Blair, Kohl, et al., 1989; Lee et al., 2012). Resting blood pressure was recorded as the first and fifth Korotkof sounds by ausculatatory methods. Serum samples were analyzed for lipids and glucose by a laboratory that participates in the CDC Lipid Standardization Program and meets its quality control standards.

Statistical Analyses

All analyses were performed using SAS® (version 9.3, Cary, NC). Frequencies or means and standard deviations were calculated for demographic and health-related characteristics; chi-square or t-tests were used to examine differences between low and high weight discrepancy at baseline. Cox proportional hazards regression was used to estimate hazard ratios (HRs) and 95% confidence intervals (95%CI) which represent the hazard (i.e., chance) of MetSyn incidence among those with high weight discrepancy, compared to those with low discrepancy. Follow-up time was the time between the baseline visit and censorship (i.e., last examination or first MetSyn ascertainment). The first model was an *a prior* model and adjusted for age, sex, physical inactivity (inactive vs. active as defined by no leisure-time activity during the three months prior to baseline), smoking status (current vs. non-smoker), alcohol intake (heavy vs. non-heavy drinker defined as > 14 drinks for men or > 7 drinks for women per week), and family history of diabetes at baseline (Wirth et al., 2014). The variable selection model was based on a backward elimination procedure. Variable selections began with a series of bi-variable analyses (i.e., weight discrepancy + covariate). If a covariate had a $p \leq .20$, it was added to the full model. Backward elimination procedures were then used to develop the final models, which included all covariates that when removed led to a 10% change in the hazard ratio (HR) of weight discrepancy or were statistically significant. The last model was developed by adding BMI to the variable selection models. All models were stratified by sex. For secondary analyses, follow-up began at time point two until censorship, which had to be at least two years. Cox proportional hazards models were applied in the same manner for the secondary analyses as described above.

Results

Average participant (n = 6,168) follow-up time was 4.8 ± 3.8 years with 1,055 (17%) newly reported MetSyn cases. At baseline, those with high weight discrepancy compared to low were more likely to be smokers (11% vs. 8%, $\chi^2 = 15.5$, $p < .01$), inactive (21% vs. 15%, $\chi^2 = 41.9$, $p < .01$), younger (mean age: 45.9 vs. 47.0, $t = 4.6$, $p < .01$), overweight (mean BMI: 26.8 vs. 23.5kg/m^2, $t = -53.2$, $p < .01$), and to have lower treadmill time (mean minutes: 18.7 vs. 21.1, $t = 20.7$, $p < .01$). Weight discrepancy between males and females was significantly different (3.9 ± 4.5 vs 5.1 ± 5.4kg, $t = -7.1$ $p < .01$, respectively); hence, sex-specific weight discrepancy medians were created.

The log-log survival curves were parallel, which indicated proportional hazards. The primary analyses examined weight discrepancy at baseline only. Among all participants, the *a priori* model (HR = 2.31, 95%CI = 2.03–2.63), variable selection model (HR = 1.96, 95%CI = 1.70–2.26), and variable selection model plus BMI (HR = 1.22, 95%CI = 1.04–1.42) indicated greater hazards for MetSyn among those with high weight discrepancy compared to those with low weight discrepancy. Statistical significance was similar between males and females (Table 1). However, larger magnitudes of effect (i.e., higher HRs) were consistently observed among females compared to males.

Secondary analyses examined the change in weight discrepancy over time. These analyses included 1,553 participants with an average of 3.4 ± 1.8 years between baseline and time point two and an average follow-up time after time point two of 5.7 ± 3.3 years. The number of participants who maintained low weight discrepancy, became low, became high, or maintained high weight discrepancy was 662 (43%), 218 (14%), 226 (15%), and 447 (29%), respectively. Those who maintained high weight discrepancy were consistently more likely to develop MetSyn compared with those who maintained low weight discrepancy for the *a priori* (HR = 3.30, 95%CI = 2.37–4.59), variable selection (HR=3.48, 95%CI = 2.49–4.86), and variable selection plus BMI models (HR = 3.44; 95%CI = 2.46–4.82). *Post hoc* comparisons indicated that those who changed to low weight discrepancy or those who changed to high weight discrepancy had elevated hazard ratios for MetSyn. However, not all were statistically significant.

Discussion

Participants who maintained high weight discrepancy over time were more likely to develop MetSyn compared with those who maintained low weight discrepancy, even after adjustment for BMI. These results are similar to our previous findings showing that those who stayed high for weight discrepancy had ≈200% greater diabetes risk compared to those who stayed low (Wirth et al., 2014). Literature corroborates our findings that individuals who have a low weight discrepancy or are weight satisfied, a potentially similar construct, are more likely to engage in healthy behaviors (e.g., physical activity, healthy eating) (Blake et al., 2013; Kuk et al., 2009).

It is possible that persistence of high weight discrepancy over time may perpetuate poor dietary (e.g., high consumption of fat, protein, sugar) and lifestyle habits (e.g., physical

inactivity, tobacco use, alcohol consumption) (Wirth et al., 2014) through mechanisms partially driven by the emotional stress and negative affect associated with these constructs (Warren, Holland, Billings, & Parker, 2012), a concept adopted from the weight dissatisfaction literature (Cernelic-Bizjak & Jenko-Praznikar, 2014). Increased stress and negative emotions associated with these constructs may influence metabolic or inflammatory processes and lead to adverse behavioral changes, which can influence health and potentially lead to MetSyn (Ahluwalia, Andreeva, Kesse-Guyot, & Hercberg, 2013; Lavie, Church, Milani, & Earnest, 2011; Pace & Miller, 2009). This hypothesis is partially confirmed by baseline findings indicating that those who had a high weight discrepancy were more likely to smoke and be inactive. Those who maintained high weight discrepancy over time were more likely to stay, or become, inactive (17% vs. 10%, $\chi^2 = 20.1$, $p < .01$) and increase their BMI (mean BMI change: +0.28 vs. −0.02 kg/m^2, $t = -4.7$, $p < .01$) compared to those who maintained low weight discrepancy. Additionally, it appears that the weight discrepancy can influence disease risk independent of obesity (i.e., adjustment for BMI). Similar findings were reported for diabetes (Wirth et al., 2014). These findings are similar to those reported for weight dissatisfaction and inflammation. A study by Cernelic-Bizjak and Jenko-Praznikar found an association between weight dissatisfaction and increased c-reactive protein and tumor necrosis factor-α regardless of obesity status (Cernelic-Bizjak & Jenko-Praznikar, 2014).

This study benefitted from a large sample size and detailed information on both measured and self-identified good weight. This analysis took advantage of a novel measure that incorporated both a subjective and objective measure of weight. Simple self-reporting level of weight satisfaction/dissatisfaction may capture other aspects of body dissatisfaction that are more susceptible to social desirability or are not specific to weight (e.g., muscle definition) (Al Sabbah, Vereecken, Abdeen, Coats, & Maes, 2009). The prospective nature of this study and exclusion of MetSyn at baseline allowed us to examine the temporality of this relationship. Limitations include the primarily European-American, middle-upper income population. Without validation analyses, which were not possible in the current study, it is difficult to establish whether weight discrepancy is a surrogate for weight dissatisfaction. It is unclear why elevated HRs for those who changed to high weight discrepancy compared to those who maintained low weight discrepancy did not achieve statistical significance. The use of a median split to characterize high versus low weight discrepancy is somewhat arbitrary. It is possible that other population groups (e.g., African Americans) may have different cultural views on body image and size (Padgett & Biro, 2003). It is possible that weight discrepancy may lead to different behaviors and outcomes in these populations. The fact that the population used in this analysis is at lower risk for cardio-metabolic disorders serves to highlight the importance of examining this relationship in populations at higher risk of MetSyn. Unfortunately, we could not adjust for social desirability, depression, or stress, either because measures for these were not collected or data were sparse.

Conclusions

This study demonstrated that greater weight discrepancy was associated with increased MetSyn risk. This is disconcerting considering that MetSyn has been associated with life-

threatening disorders, including CVD (Grundy, 2008; Grundy et al., 2004). Future research should continue to elucidate the effect of weight discrepancy on chronic weight-related disorders and examine its utility as an indicator for risk in screening for MetSyn. Furthermore, weight discrepancy could be used to refine targeting of intervention efforts. The emphasis on weight loss in many health promotion programs for chronic disease prevention may not include messages that are salient to those with large weight discrepancies. These findings suggest that future interventions targeting weight-related chronic disorders should incorporate components to address weight discrepancies and focus on understanding what factors motivate those who have a large weight discrepancy to engage in positive health behaviors which could reduce risk of chronic disorders.

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Highlights

- Weight discrepancy was defined as measured minus self-reported goal weight.
- Weight discrepancy was associated with smoking and physical inactivity at baseline.
- Chronic high weight discrepancy was associated with metabolic syndrome risk.
- Interventions targeting weight discrepancy may help lower metabolic syndrome risk.

Table 1

Hazard Ratios and 95% Confidence Intervals for Metabolic Syndrome Incidence among All Participants and Stratified by Sex

Weight Discrepancy: High vs. Low	All Participants (*n* = 6,168)		Males (*n* = 5,183)		Females (*n* = 985)	
	HR	**95% CI**	**HR**	**95% CI**	**HR**	**95% CI**
Crude model	2.28	2.01–2.59	2.16	1.90–2.46	5.01	2.61–9.59
a priori model[a]	2.31	2.03–2.63	2.19	1.92–2.50	4.66	2.40–9.04
Variable selection model[b]	1.96	1.70–2.26	1.54	1.33–1.79	2.41	1.14–5.08
Variable selection model + BMI[c]	1.22	1.04–1.42	1.17	1.00–1.37	1.91	0.89–4.18

[a] Adjusted for physical activity, smoking status, alcohol consumption, family history of diabetes, and age.

[b] Adjusted for family history of diabetes, age, year of examination, body fat percentage, and treadmill test minutes.

[c] Same adjustments as Model ‘b’ plus body mass index.

High and low weight discrepancies were based on median splits for males and females separately.

Abbreviations: HR = hazard ratio; 95% CI = 95% Confidence Interval; BMI = body mass index (kg/m^2).

Table 2

Hazard Ratios and 95% Confidence Intervals for Metabolic Syndrome Incidence for the Change in Weight Discrepancy

Weight Discrepancy Change	All Participants (*n* = 1,553)	
	HR	**95% CI**
Crude model		
Maintained Low	1.00	Referent
Became Low	1.85	1.18–2.90
Became High	1.39	0.86–2.27
Maintained High	3.30	2.37–4.59
***a priori* model**[a]		
Maintained Low	1.00	Referent
Became Low	1.91	1.22–3.00
Became High	1.46	0.89–2.37
Maintained High	3.47	2.48–4.86
Variable selection model[b]		
Maintained Low	1.00	Referent
Became Low	1.78	1.13–2.82
Became High	1.58	0.96–2.59
Maintained High	3.48	2.49–4.86
Variable selection model + BMI[c]		
Maintained Low	1.00	Referent
Became Low	1.84	1.16–2.92
Became High	1.51	0.91–2.51
Maintained High	3.44	2.46–4.82

Estimates could not be obtained for females due to small sample sizes and therefore, only results for all participants are displayed.

[a] Adjusted for baseline age and family history of diabetes and the difference in physical activity, smoking status, and alcohol intake between baseline and time point 2.

[b] Adjusted for baseline age and the difference in body fat percentage.

[c] Same model as model 'b' plus adjustment for the difference in BMI.

Abbreviations: HR = hazard ratio; 95% CI = 95% Confidence Interval; BMI = body mass index (kg/m^2).